Epitomes

Important Advances in Clinical Medicine

Internal Medicine

The Scientific Board of the California Medical Association presents the following inventory of items of progress in internal medicine. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, researchers, or scholars to stay abreast of these items of progress in internal medicine that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Internal Medicine of the California Medical Association, and the summaries were prepared under its direction.

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Ventilator Management of Severe Asthma

ACUTE, SEVERE, POTENTIALLY life-threatening asthma is recognized to have several features that stress the predominance of bronchial mucosal inflammation and edema and debris in the airway lumen, in conjunction with bronchial smooth muscle contraction, as the causes of airway obstruction. One important clinical consequence is that the increasingly severe airway obstruction may show marked diurnal fluctuation. Other features, results of the predominance of airway inflammation compared with bronchospasm, include prolonged severe bronchial lumen narrowing once established, refractoriness to bronchodilators, and the need for therapy that addresses the inflammatory component.

When acute severe asthma develops, the mainstays of therapy are corticosteroids plus bronchodilators, including inhaled β-adrenergic agonists, and, to a lesser extent, theophylline and ipratropium bromide. The beneficial effect of corticosteroids requires at least several hours.

The slow response of airway inflammation to pharmacologic treatment means that even with aggressive medical therapy, respiratory failure requiring mechanical ventilation develops in some asthmatic patients. Because of the greatly increased work of breathing due to increased airway resistance, these patients cannot maintain sufficient ventilation to compensate for increased ventilation-perfusion mismatching. Mechanical ventilation, however, has been associated with especially high mortality rates in asthmatic patients (22% in one recent series), and serious complications of barotrauma and impaired cardiovascular function are not infrequent.

Recent studies have emphasized the likelihood of hyperinflation in asthmatic patients who are mechanically ventilated. The narrowed airways in asthmatic patients have a more pronounced effect on expiratory than on inspiratory resistance; this is true whether patients are spontaneously breathing or mechanically ventilated. If there is insufficient time for exhalation, air trapping or hyperinflation may result. Hyperinflation has a major effect on gas exchange, especially increasing the physiologic dead space, and may result in tidal ventilation at a less compliant portion of the respiratory system pressure-volume curve. Thus, asthmatic patients in whom hyperinflation develops during mechanical ventilation may have worsening of gas exchange and require dangerously high airway pressures.

New strategies for mechanical ventilation in patients with acute asthma emphasize the use of high inspiratory flow and slow breathing frequency—the latter sometimes requiring sedation or neuromuscular blockade—to prolong expiratory time and allow end-expiratory lung volume to decrease to more normal levels. An improvement in arterial blood gas values, at times paradoxically in response to decreased minute ventilation, or a fall in peak airway pressure is indicative of decreased hyperinflation. In many patients, hyperinflation may be identified by the presence of "intrinsic positive end-expiratory pressure"—most easily detected by finding positive airway pressure during occlusion of the exhaust port of the ventilator immediately before the next inspiration—and this finding should be used to guide changes in ventilatory settings. The administration of a high inspired concentration of oxygen (40% to 50% of oxygen) may help inhibit hypoxic respiratory drive, slowing the respiratory rate and prolonging the expiratory phase. Some investigators have suggested that some degree of hypercapnia can be accepted as a consequence of limiting airway pressure, tidal volume, or respiratory frequency, rather than using more conventional ventilatory settings intended to provide a patient with sufficient ventilation to maintain normal P_{CO_2} and pH. This "controlled hypoventilation" may result in reduced complications from hyperinflation.

The use of more recently introduced modes of ventilation, such as pressure support and pressure control, has yet to be compared with conventional modes in clinical trials in asthmatic patients. Both of these modes may be used to limit peak airway pressure, but other characteristics may or may not be optimal for use in acute airway obstruction.

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